Tracking how flu evolves _ it has sticky tricks

LAURAN NEERGAARD - AP Medical Writer - Associated Press

Vaccinating more children might help slow the evolution of the constantly changing flu virus, government scientists reported Thursday.

Influenza is a mighty mutator. Sometimes it makes big changes that result in never-before-seen strains, like this year's swine flu. But from season to season, it undergoes subtle shape-shifting, which is the reason people need a new winter flu vaccine every year.

This regular shift is what's called antigenic drift. Think of influenza as wearing a coat, a protein on the virus' surface — the "H'' in the family flu names, for hemagglutinin — that is the major factor in triggering the immune system to mount an attack. Slight changes in the appearance of that coat can be enough to confuse your immune system and allow infection.

But just how do those changes happen? National Institutes of Health researchers infected lots of mice — vaccinated and unvaccinated ones — to watch that evolution in action. They report in the journal Science that influenza responds to immune pressure, a discovery that supports today's vaccine policy urging more children to get their flu shots each fall.

Even a vaccine that isn't a perfect match to the virus may provide some protection and limit the scope of changes in the virus.

"We're giving the virus more wiggle room, more evolutionary space, by having some naive individuals," explained lead researcher Dr. Jonathan Yewdell of NIH's National Institute of Allergy and Infectious Diseases. To scientists, "naive" means the previously unexposed.

Yewdell's team took an old strain of seasonal H1N1 flu that circulated decades ago and infected groups of mice. After each infection, they culled virus from mouse lungs and used that to infect more sets of mice. After nine such cycles, they checked the hemagglutinin gene for mutations. Unvaccinated mice had none. In vaccinated mice, the virus had mutated to become stickier — it changed the way it binds to cells to clamp on more tightly, which helps it evade an attack from immune cells called antibodies.

But that's a big tradeoff: A stickier virus doesn't spread as easily, Yewdell said.

Then the team took the new sticky mutant and infected more unvaccinated mice with it — and here's the surprise: The virus mutated back into a less sticky, easier-to-spread version.

That's why there are implications for people, because children who haven't yet

caught or been vaccinated against many flu strains would offer a similar opportunity for mutation to easier-to-spread versions.

"We want to box flu in as much as we can," Yewdell said. "With more antibody pressure (from vaccinated people), it's got to bind tighter and the virus is not so happy."

In a separate study published in Science, a team led by University of Georgia researchers examined ponies vaccinated against equine influenza to determine how much antigenic drift it takes to outwit a particular year's flu vaccine. It's a model that scientists might use to track a worsening outbreak. But the take-home message — if enough of a population is vaccinated, even an imperfect vaccine can provide some benefit — is potentially useful information the next time a worrisome new strain crops up.

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